tropenia associated with anorexia nervosa, marrow reserves are adequate and there is no increased incidence of associated opportunistic infections.[17]

Another possible cause of pulmonary injury in malnourished patients involves an associated antioxidant deficiency. Glutathione and other similar proteins are depleted in malnourished patients. In animal studies, increased susceptibility to oxygen toxicity occurs with starvation, especially at the oxygen concentrations required to oxygenate our patient.[18,19]

Several behaviors associated with anorexia nervosa and bulimia predispose patients to pulmonary complications. Pneumomediastinum has been reported in association with bulimia.[20] Aspiration of foreign material is also a major concern. Though mentioned in reviews, there are no reports documenting this complication.[4] Self-mutilative behavior without suicidal intent, in addition to alcohol and drug abuse, is frequently seen in bulimic patients.[21] The circumstances surrounding our patient's lipoid pneumonitis suggest a volitional component. The use of mineral oil as a laxative with accidental inhalation or aspiration is also possible.

Exogenous lipoid pneumonia is an unusual pulmonary problem and has not been reported previously in association with anorexia nervosa.[22] Though most often found as a slowly progressive, minimally symptomatic disease process, it occasionally presents as an acute pneumonitis.[23,24] There is a high incidence of secondary infection due to plugging and impaired mucociliary clearance.[25,26] Occasionally the pneumonitis progresses to interstitial fibrosis.[27] Respiratory failure is rare. Several factors mentioned previously may have contributed to this patient's pulmonary disease, although pathologically no other cause could be found. The use of corticosteroids has been suggested as a potential treatment.[28] With corticosteroid treatment, our patient has shown moderate improvement in lung function, suggesting resolution of the acute pneumonitis with residual fibrosis.

Pulmonary disorders in patients with anorexia nervosa are uncommon, though multiple complications can occur. With the frequent abuse of laxatives, including mineral oil, by anorexic patients, lipoid pneumonia may develop more commonly than previously appreciated. The insidious nature of lipoid pneumonia makes this an easily overlooked and potentially severe complication, which requires a high index of suspicion for prompt diagnosis.

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Coexistent Dermatomyositis and Autoimmune Thyroiditis

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DERMATOMYOSITIS is a disease characterized by nonsuppurative inflammation of skeletal muscle and characteristic skin changes. While dermatomyositis-like syndromes due to hypothyroidism have been well described, the occurrence of both hypothyroidism due to autoimmune thyroiditis and dermatomyositis together is not well documented.

We describe the case of a patient with coexistent autoimmune thyroiditis and dermatomyositis, showing clearly that these diseases can coexist. He was treated with pharmacologic doses of both steroids and thyroid hormone and is in clinical remission after two years.

Report of a Case

The patient, a 50-year-old male rice farmer, presented with proximal muscle weakness, Raynaud's phenomenon, dysphagia, hoarseness, and loss of energy for six months. On physical examination he had profound muscle weakness in the limb girdle and neck flexors. The patient was unable to lift his head from the pillow, rise from a chair, or walk up stairs without assistance. No fasciculations were seen. The neurologic examination elicited no abnormalities. Examination of the neck showed no goiter. His skin was remarkable for pronounced periungual erythema, a heliotrope rash over the upper eyelids, and Gottron's papules on the knuckles.

Admission laboratory tests were remarkable for the fol-

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ABBREVIATIONS USED IN TEXT
CK = creatine kinase
EMG = electromyogram/phic
TSH = thyroid-stimulating hormone

lowing values: creatine kinase (CK) 3,172 units per liter (normal 35 to 232); serum aspartate aminotransferase (glutamic-oxaloacetic transaminase) 112 units per liter (normal 0 to 41); lactate dehydrogenase 535 units per liter (normal 60 to 200); aldolase 28 mU per ml (normal 0.5 to 3.1); thyroxine 2.3 μg per dl (normal 5 to 12); free thyroxine index 0.9 (normal 1.7 to 5.4); thyroid-stimulating hormone (TSH) 45.1 IU per ml (normal 0.45 to 6.2); antinuclear antibody positive at 1:1,280 in a nucleolar pattern; antithyroid microsomal antibody positive at 1:1,600 dilution (normal less than 1:100); and antithyroglobulin antibody positive at 1:160 (normal less than 1:10).

A thyroid scan using radioactive iodine showed diffusely low uptake at five hours of 4.2% (normal 5% to 15%) and 24 hours' uptake at 10.6% (normal 12% to 36%). The thyroid size was estimated to be 20 grams. An erythrocyte sedimentation rate, chest x-ray film, electrocardiogram, complete blood count, anti-Smith antibody, anti-double-stranded DNA antibody, complement levels of C3 and C4, and antiribonuclear protein were all negative or normal. The test for the polymyositis-1 antigen was not done. An electromyogram (EMG) was diffusely abnormal with fibrillations, insertional irritability, positive sharp waves, and short-duration action potentials consistent with inflammatory myopathy. A biopsy specimen of the quadriceps showed perifascicular atrophy with lymphatic infiltrations of perifascicular collagen, muscle fascicle necrosis of type 1 and type 2 fibers, and basophilia consistent with polymyositis. An occult carcinoma workup was negative.

Clinical Course

Therapy consisting of the administration of levothyroxine sodium (Synthroid), 0.15 mg per day, and prednisone, 60 mg per day, was started. The CK level came down to 224 units per liter at one month and the TSH was down to 4.1 IU per ml. The prednisone dosage was rapidly tapered to 20 mg per day and at three months the CK value had returned to normal.

Clinically, the patient had quick resolution of his dysphagia, but muscle strength improved only slowly. Attempts to wean the prednisone therapy completely have been unsuccessful, and he is currently maintained on a regimen of 7.5 mg per day. At a two-year follow-up visit, his CK level and thyroid function tests remain normal, and muscle strength is adequate for farming chores.

Discussion

Hypothyroidism has long been associated with muscle weakness. Several authors have described a hypothyroid state clinically mimicking polymyositis/dermatomyositis.[1-6] Differentiating these diseases can be quite difficult, often relying on clinical grounds because laboratory test results can be ambiguous. We are aware of only one case report associating dermatomyositis and autoimmune thyroiditis.[7] Our patient presented with the clinical features of dermatomyositis and was found to have coexistent hypothyroidism due to autoimmune thyroiditis.

This case shows the difficulties of interpreting laboratory data to diagnose these diseases. Muscle enzyme activity can be normal or elevated in both diseases.[8-10] Similarly, EMG studies in hypothyroid patients with myopathy have had a wide variety of findings, from fibrillations and positive sharp waves to normal results,[8,10-14] although recent authors tend to support the view that the EMG should be normal in patients with hypothyroidism.[13] Muscle biopsy specimens from hypothyroid patients have shown such diverse features as lymphocytic infiltrates, basophilia, and focal necrosis,[1,14,15] typically seen in patients with polymyositis, while others have had no pathologic findings.[15(pp642-643)] One conceivable explanation of these ambiguities lies in the possibility that some of those hypothyroid patients with abnormal EMG and muscle biopsy findings may have both diseases coexisting.

Immunologic studies have shown that both of these diseases are autoimmune mediated. Suppressor T-cell dysfunction and lymphocyte-mediated cytotoxicity have been well described in cases of polymyositis,[16] and there is evidence of suppressor T-cell dysfunction in autoimmune thyroiditis as well,[17-19] suggesting a common pathogenesis. In addition, the incidence of histocompatibility antigens HLA-B8 and DR3 is increased in frequency in autoimmune thyroiditis[20] and adult dermatomyositis.[21] In our patient and in that of White and Tesar,[7] hypothyroidism due to autoimmune thyroiditis most likely preceded the onset of dermatomyositis.

In conclusion, it is important for clinicians to be more aware that dermatomyositis and hypothyroidism due to autoimmune thyroiditis can coexist and a thorough evaluation of both may be necessary in patients presenting with proximal muscle weakness. In addition, further studies of the immunologic aspects of both diseases may unravel common pathways of autoimmune-mediated cellular dysfunction.

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